

## Effect of Kerosene on the Respiratory Organ (Gill) of African Catfish (*Clarias gariepinus*)

Jacob Eriti Abel<sup>1</sup>, Akwaowo D. Denny<sup>2</sup>, Victor Eyo<sup>3</sup>

<sup>1&3</sup>Department of Fisheries and Aquaculture, Faculty of Oceanography, University of Calabar, Calabar, Nigeria.

<sup>2</sup>Department of Environmental Management, Faculty of Environmental Science, University of Calabar, Calabar, Nigeria.

<sup>1</sup>Corresponding author's Email address: [eritijacob1998@gmail.com](mailto:eritijacob1998@gmail.com)

### Abstract

*This study investigated the effect of kerosene on the respiratory organ (gill) of juvenile African catfish (Clarias gariepinus) under laboratory condition for 96hours. A total of hundred and twenty (120) juvenile C. gariepinus was used in six (6) different aquaria having duplicates i.e. ten (10) juveniles each were grouped into twelve (12) different test aquaria and held for 24, 48, 72 and 96 hours in six (6) different concentrations of kerosene (0, 0.4, 0.8, 1.2, 1.6, 2.0ml/L). At the end of the test period, histopathological examination of the gills was conducted. The LC50 of kerosene in the water was noted to be 1.05ml/L. During the period of exposure of test fish to this toxicant, respiratory distress, aggression, weakness, erratic swimming, loss of balance, sluggish movement were common reactions of the test fish at the time of toxicant application before death. It was also observed that mortality was toxicant dependent (i.e. the higher the concentration of toxicant the higher the number of dead fish recorded). Histopathological examination of the gills exposed to kerosene showed lesions, degradation of filament, and necrosis with increasing concentration of kerosene, which in relation to previous studies indicates reduced dissolved oxygen level, resulting in respiratory challenge/difficulty. All the juveniles held in the control aquaria showed no histopathological degradation. Conclusively, this study has been able to reveal that exposing juvenile African catfish to even low concentrations of kerosene could lead to histopathological damage on the fish gill structure. Conclusively, a safe concentration was established using an application factor of 0.01 (for industrial chemicals and pesticides). We arrived at safe concentration level of 0.0105 ml/L. Thus, if the kerosene concentrations remain below 0.0105 ml/L, juveniles of C. gariepinus as well as other aquatic species will suffer no adverse effects.*

**Keywords:** Kerosene; *Clarias gariepinus*; Respiratory organ; histopathological effect; Nigeria.

### 1.0 Introduction

Kerosene is a refined petroleum product comprising hydrocarbon mixtures with carbon chains ranging from 11 to 18 atoms per molecule (Ekejiuba, 2021). Its versatility makes it widely used as fuel for automobiles, generators, heating, cooking, and aviation, as well as in industrial applications such as paint and grease production (Kakodia et al, 2024). However, the increasing reliance on petroleum products has led to growing environmental concerns, particularly regarding their effects on aquatic ecosystems (Ukhurebor et al., 2021; Adeola et al., 2022). Oil spills, pipeline corrosion, vandalization, and accidental discharges are among the primary causes of oil pollution, introducing hydrocarbons like kerosene into freshwater and marine environments (Edori et al., 2014). Crude oil and its refined products, including kerosene, pose significant threats to aquatic ecosystems (Shah and Soni, 2024). They reduce dissolved oxygen availability, disrupt biochemical and physiological

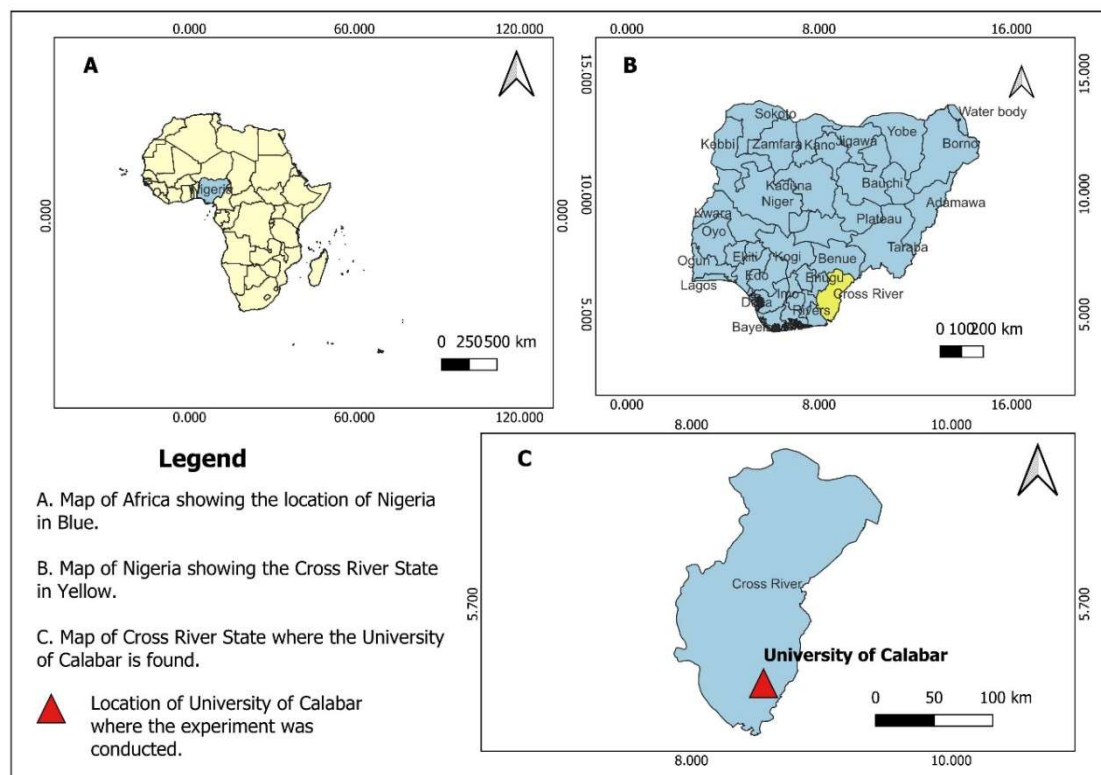
activities of aquatic organisms, and accumulate in food chains, resulting in long-term ecological damage (Kakodia et al., 2024). These pollutants often impact fish, which serve as bioindicators due to their sensitivity to changes in water quality (Ivon et al., 2021). Behavioral, haematological, and histopathological alterations in fish exposed to pollutants are frequently used as biomarkers to assess contamination levels (Eseigbe et al., 2013). Despite the wealth of studies on crude oil toxicity, limited research has focused specifically on kerosene's toxic effects on fish health and physiology (Adeola et al., 2022). Earlier studies have demonstrated that water-soluble fractions of crude oil impair fish growth, induce physiological stress, and affect survival (Santos et al., 2022). These findings underscore the need for further investigation into the specific concentration thresholds at which petroleum fractions become toxic to aquatic organisms (Chukwu & Okhumale, 2009).

In Nigeria's Niger Delta region, petroleum exploration has contributed significantly to national wealth but has also resulted in widespread environmental degradation (Bamidele and Eramah, 2023). Oil spills, operational discharges, and sabotage of petroleum infrastructure have led to pollution of aquatic ecosystems, threatening biodiversity and regional fisheries (Nwilo et al., 2006). The release of hydrocarbons such as kerosene into water bodies often occurs through runoff, erosion, and accidental discharges during transportation and storage, exacerbating contamination levels (Kanungo et al., 2024 Scheren et al., 2002; Adam et al., 2002). Additionally, environmental contamination by petroleum products is particularly concerning because of its persistence and long-term impacts. While hydrocarbons in kerosene undergo photodegradation in air and biodegradation in soil and water, the initial exposure can cause irreversible harm to aquatic life (Pauline, 2006). Fish, being intimately connected to their aquatic environment, are particularly vulnerable to such pollution. For instance, petroleum products disrupt oxygen exchange at the gills, impair metabolic processes, and accumulate in tissues, leading to physiological stress and mortality (Nwamba et al., 2006). The ecological and socio-economic implications of kerosene pollution are far-reaching (Bashir, 2021). Fish mortality and population decline disrupt fisheries, which are critical for food security and livelihoods in developing countries (Daniel-Kalio et al., 2002). Additionally, contamination of aquatic ecosystems reduces water quality, impacting other organisms and human populations that depend on these resources. Given the importance of fish such as *C. gariepinus* in aquaculture and the broader ecosystem, understanding the toxicological effects of kerosene is imperative. Several studies have been conducted on the exposure of *C. gariepinus* to various pollutants. Abdel-Moneim et al. (2008) conducted a study on the physiological and histopathological effects of juvenile catfish exposed to dyestuff and chemical waste water. Doherty et al. (2013) conducted another study on the toxicological effect and histopathology of *C. gariepinus* exposed to water soluble fractions of Diesel and kerosene. George et al. (2014) investigated the acute toxic effect of Qua Iboe light crude oil on the gills of *C. gariepinus* juveniles. Esie (2018) also conducted a study on the effects of produced water on juvenile *C. gariepinus*. Additionally, Edori et al. (2014) conducted a study on the comparative toxicity of petrol and kerosene to periwinkle (*Tympanotonus fuscatus*). Moreover, Gabriel et al. (2007), conducted a study on the haematology and gill pathology of *C. gariepinus* exposed to refined petroleum oil, kerosene under laboratory conditions, amongst other studies. However, these studies have never investigated the effect of kerosene fraction of crude oil on the respiratory organ (gills) of *C. gariepinus* a key species in Nigerian aquaculture industry. Therefore, the main aim this study was to determine the effects of kerosene on the respiratory organ (gills) of juvenile *C. Gariepinus* by specifically looking at a) To determine the LC50 of kerosene on the *C. Gariepinus* b) to determine the safe concentration of kerosene on *C. gariepinus* c) to determine the histopathological effect of kerosene on *C. gariepinus* and d) to check behavioural changes in the *C. gariepinus*.

## **2.0 Materials and methods**

## 2.1 Study area

This study was conducted at the University of Calabar, Faculty of Oceanography in the Laboratory of Fisheries and Aquaculture Laboratory (Figure 1).



**Fig 1:** Map showing the location of University of Calabar where the experiment was done.

## 2.2 Specimen collection and acclimatization

A total of 100 healthy juveniles of *C. gariepinus* of mean weight ( $1.31 \pm 0.56$  g) and mean total length ( $5.62 \pm 0.61$  cm) were purchased from University of Calabar fish farm and hatchery complex located at Latitude 04°05'02"N and Longitude 008°02'45"E respectively (Akpan et al., 2002). The fish was transported to Fisheries and Aquaculture Laboratory, Faculty of Oceanography, University of Calabar with the aid of 20 Liters plastic container. The fish was kept in 20 Liters plastic tanks and acclimatized to laboratory conditions for a period of two weeks prior to the start of the experiment. During acclimatization, the fish was fed with 2 mm Coppens fish feed containing 45% crude protein (Table 1) at 5 % of their body weight twice daily, between the hours of 8:00-9:00hr and 17:00-18:00 pm. Water quality was managed by changing of water in the aquaria daily between the hours of 6am-7am.

**Table 1: Composition of Coppens feed**

| Composition   | Percentage (%) |
|---------------|----------------|
| Crude protein | 45             |
| Ash           | 9.55           |
| Crude fibre   | 12             |

|             |     |
|-------------|-----|
| Crude fat   | 1.5 |
| Phosphorous | 2   |

Source: Manufacturer

## 2.2 Experimental design

### 2.2.1 Collection of Kerosene

Five (5) litres of kerosene used for this experimental study was purchased from the Fynefield Plc, located at Goldie Road, Calabar, Cross River State.

### 2.2.2 Range finding test

After the period of acclimatization, a preliminary range finding test was conducted following standard methods of APHA (2005) to determine the concentrations that was used in the actual experiment. Ten (10) fish each was placed in five different exposure chambers having five (5) litres of water each with replicate having a broad range of concentrations (0, 1, 2, 3, and 4 ml/l). Survival and mortality were recorded after 1 and 24 hours and the results were used to determine the definitive test concentrations.

### 2.2.3 Acute Toxicity Test

After the range finding test, six concentrations of kerosene were used to conduct acute toxicity test under standard bioassay procedure. Mortality was monitored for 96 hours. Any fish that fails to move its body was classified “dead”. Floating or sunk fish which do not move was brought out and classified dead using a pair of gloves and recorded.

### 2.2.4 96 Hours Lethal Concentration (LC50)

The 96-hour LC50 was estimated using the methods recommended by UNEP (1989).

### 2.2.5 Measurement of water quality parameters

pH, temperature and dissolved oxygen was monitored during the test period according to standard method of the American Public Health Association (APHA, 1989). pH level was measured using pH meter (pH Model SAEG pHS-25C. Temperature was measured using thermometer (mercury in glass thermometer). Dissolved oxygen level was measured using DO meter (MW600 Dissolved oxygen Milwaukee Smart DO meter).

## 2.3 Physicochemical analysis of kerosene

The physicochemical analysis of the kerosene used in this study was done using standard American water was carried out using standard American Public Health Association (APHA) methods

### 2.3.1 Odour and colour

The odour and colour of kerosene used in this study was determined using physical observation with the nose and eye respectively.

## 2.4 Monitoring of specimen for death rate

Any animal (fish) that fails to move its body was classified “dead. Floating or sunk fish which do not move was brought out and classified dead using a pair of gloves and recorded.

## 2.5 Histopathology of the gills

The gills were extracted from the test fish at each concentration and processed manually following the stepwise procedure described by Bancroft and Cook (1994). Initially, the gills were fixed in 10% formalin for 48 hours and thoroughly washed with water to remove excess fixatives. The fixed tissues were dehydrated in ascending grades of ethanol (30%, 50%, 70%, 90%, and 100%) for at least two hours in each solution. Dehydrated tissues were cleared in xylene to enhance microscopic examination, starting with a 1:1 mixture of chloroform and xylene, followed by pure xylene. The tissues were then impregnated with paraffin wax, melted at 60°C, and left to infiltrate for two hours to facilitate sectioning with a microtome. The gills were embedded in molds, blocked out on wooden blocks for microtomy, and sectioned using a rotary microtome at 10 µm thickness. The sections were stained using hematoxylin and eosin methods, and photomicrographs of the stained tissues mounted on glass slides were captured digitally using an Amcap microscope camera (Model DCE-2).

### 3.0 Results

#### 3.1 Behavioural response of *C. gariepinus* exposed to kerosene

The behavioural responses observed during this study include:

- a) Loss of balance
- b) Erratic swimming
- c) Aggression
- d) Weakness
- e) Death.

#### 3.1 Mean mortality of *C. gariepinus* exposed to kerosene

A regular trend of mortality was observed with an increase in toxicant concentration (Table 2). At the early stage, (i.e. the first 24hrs) the mortality rate was so high, with progressive exposure of fish to toxicant, 48hrs, 72hrs and 96hrs mortality rate reduced drastically and fish started moving again freely. Between 72 and 96hrs, there were no survivors recorded in the highest concentration (2.0ml/L).

**Table 2: Mean mortality of *C. gariepinus* exposed to kerosene**

| Concentration | 24 hours    | 48 hours  | 72 hours  | 96 hours  | Mortality  |
|---------------|-------------|-----------|-----------|-----------|------------|
| 0.0 ml/l      | 0.00±0.00   | 0.00±0.00 | 0.00±0.00 | 0.00±0.00 | 0.00±0.00  |
| 0.4 ml/l      | 0.50 ± 0.50 | 0.00±0.00 | 0.00±0.00 | 0.00±0.00 | 0.50±0.50  |
| 0.8 ml/l      | 0.50±0.50   | 0.50±0.50 | 0.00±0.00 | 0.00±0.00 | 1.00±1.00  |
| 1.2 ml/l      | 6.50±2.50   | 0.00±0.00 | 0.00±0.00 | 0.50±0.50 | 7.00±2.00  |
| 1.6 ml/l      | 7.50±0.50   | 0.00±0.00 | 0.00±0.00 | 0.00±0.00 | 7.50±0.50  |
| 2.0 ml/l      | 10.0 ± 0.0  | 0.00±0.00 | 0.00±0.00 | 0.00±0.00 | 10.00±0.00 |

#### 3.3 Mean percentage mortality of *C. gariepinus* exposed to kerosene

Results obtained for mean percentage mortality of *C. gariepinus* exposed to kerosene (Table 3) shows that mean mortality and mean percentage mortality was 0.00 ± 0.00 and 0.00 ± 0.00 % in 0.0 ml/L. In 0.4 ml/L, mean mortality and mean percentage mortality was 0.50 ± 0.00 and 5.00 ± 0.00%. In 0.8ml/L mean mortality and mean percentage mortality was 1.00±1.00 and 10.00±10.00%. In

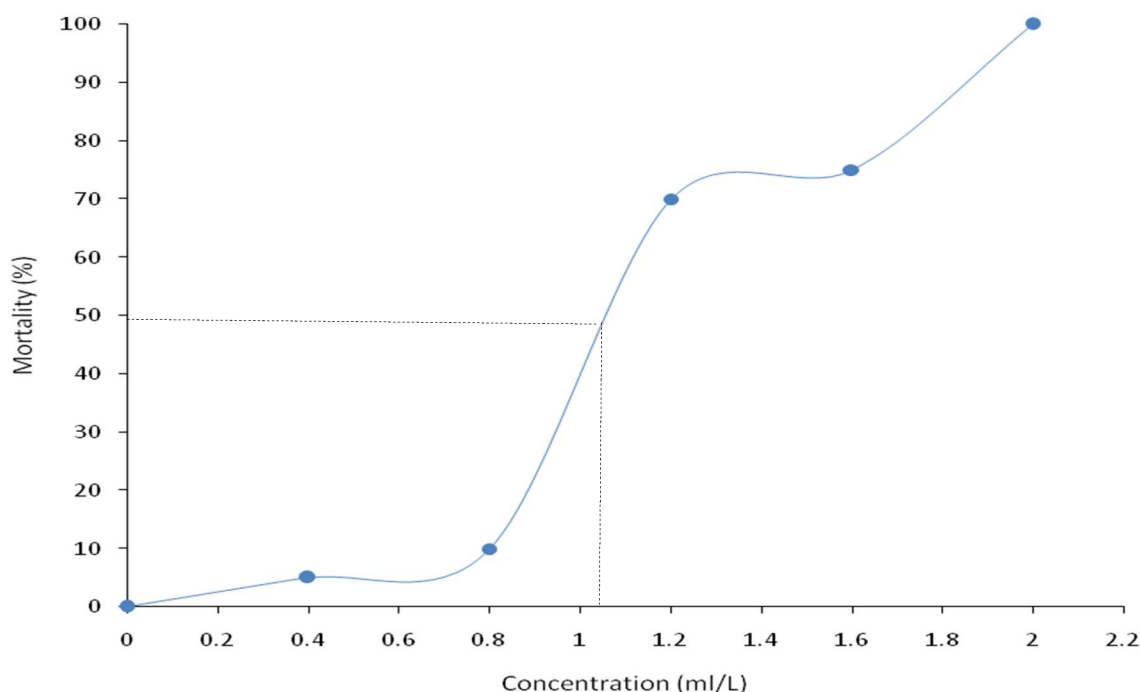
1.2ml/L mean mortality and mean percentage mortality was  $7.00 \pm 2.00$  and  $70.00 \pm 20.00\%$ . In 1.6ml/L mean mortality and mean percentage mortality was  $7.50 \pm 0.50$  and  $75.00 \pm 5.00\%$ . In 2.0ml/L mean mortality and mean percentage mortality was  $10.00 \pm 0.00$  and  $100.00 \pm 0.00\%$ .

**Table 3: Mean percentage mortality of *C. gariepinus* exposed to kerosene**

| Concentration | Mortality        | Percentage mortality (%) |
|---------------|------------------|--------------------------|
| 0.0 ml/l      | $0.00 \pm 0.00$  | $0.00 \pm 0.00$          |
| 0.4 ml/l      | $0.50 \pm 0.50$  | $5.00 \pm 5.00$          |
| 0.8 ml/l      | $1.00 \pm 1.00$  | $10.00 \pm 10.00$        |
| 1.2 ml/l      | $7.00 \pm 2.00$  | $70.00 \pm 20.00$        |
| 1.6 ml/l      | $7.50 \pm 0.50$  | $75.00 \pm 5.00$         |
| 2.0 ml/l      | $10.00 \pm 0.00$ | $100.00 \pm 0.00$        |

### 3.4 96 Hours Lethal concentration (LC50) of *C. gariepinus* exposed to kerosene

Results obtained for the 96 hours lethal concentration (LC50) of kerosene to *C. gariepinus* (Fig. 1) shows that the LC50 was 1.05 ml/L.



**Figure 2: Mortality (%) of *C. gariepinus* against concentrations of kerosene showing LC50**

### 3.5 Mean values of physico-chemical parameters measured during 96 hour exposure of kerosene to *Clarias gariepinus*

Results obtained for the mean physico-chemical parameters of the experimental aquaria (Table 4) shows that in 0.0 ml/L, pH was  $5.82 \pm 0.00$ , dissolved oxygen ( $3.60 \pm 0.00$  mg/L) and temperature ( $28.00 \pm 0.00$  °C). In 0.4 ml/L, pH was  $6.20 \pm 0.00$ , dissolved oxygen ( $3.20 \pm 0.00$  mg/L) and temperature ( $28.00 \pm 0.00$  °C). In 0.8 ml/L pH was ( $7.19 \pm 0.00$ ), dissolved oxygen was

( $3.10 \pm 0.00$ mg/L) and temperature ( $28.00 \pm 0.00$  °C). In 1.2ml/L pH was ( $6.47 \pm 0.00$ ), dissolved was ( $3.30 \pm 0.00$ mg/L), and temperature ( $28.00 \pm 0.000$ C). In 1.6ml/L pH was ( $6.09 \pm 0.00$ ), dissolved oxygen was ( $3.30 \pm 0.00$ mg/L) and temperature ( $28.00 \pm 0.000$ C). In 2.0ml/L pH was ( $6.10 \pm 0.00$ ), dissolved was ( $3.10 \pm 0.00$ mg/L) and ( $28.00 \pm 0.000$ C).

**Table 4: Mean values of physico-chemical parameters measured during 96-hour exposure of kerosene to *C. gariepinus***

| Concentration | pH              | Dissolved oxygen (mg/l) | Temperature (°C) |
|---------------|-----------------|-------------------------|------------------|
| 0.0 ml/L      | $5.82 \pm 0.00$ | $3.60 \pm 0.00$         | $28.00 \pm 0.00$ |
| 0.4 ml/L      | $6.20 \pm 0.00$ | $3.20 \pm 0.00$         | $28.00 \pm 0.00$ |
| 0.8 ml/L      | $7.19 \pm 0.00$ | $3.10 \pm 0.00$         | $28.00 \pm 0.00$ |
| 1.2 ml/L      | $6.47 \pm 0.00$ | $3.30 \pm 0.00$         | $28.00 \pm 0.00$ |
| 1.6 ml/L      | $6.09 \pm 0.00$ | $3.40 \pm 0.00$         | $28.00 \pm 0.00$ |
| 2.0 ml/L      | $6.10 \pm 0.00$ | $3.10 \pm 0.00$         | $28.00 \pm 0.00$ |

### 3.6 Histopathology of the gills of *C. gariepinus* exposed to different concentration of kerosene

Histopathology of the gills of *C. gariepinus* (Figure 3-6) exposed to different concentration of kerosene showed that in the control (0.0 ml/L), a normal gill membrane with no lesion, necrosis and inflammation was obtained. In 0.8ml/L, gills showed slight necrosis and degeneration filament. In 1.6ml/L gills showed degeneration of filament and in 2.0ml/L gills showed severe degeneration of filament, necrosis and areas of lesion.



**Figure 3:** Control (Arrow shows normal gill membrane with no lesion, necrosis and inflammation)

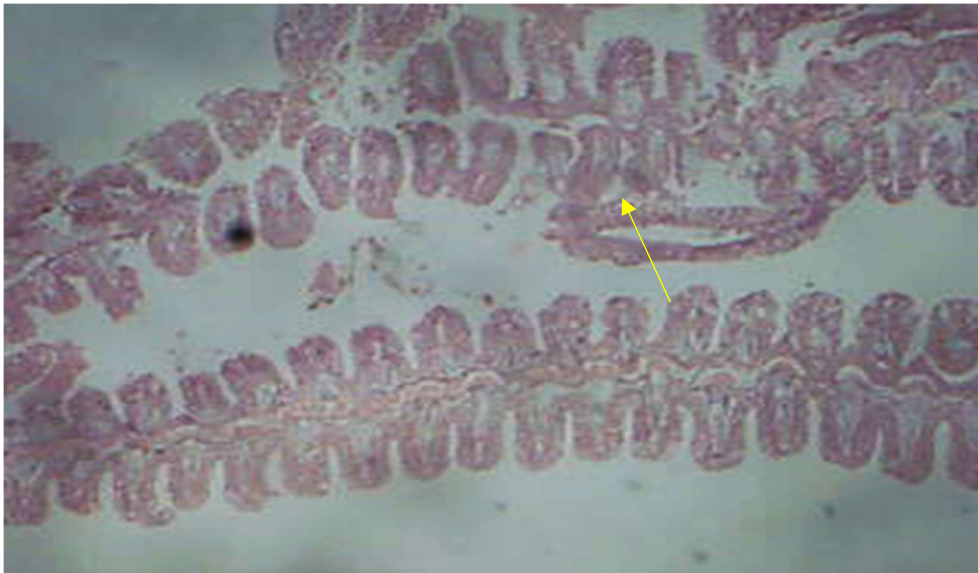


Figure 4 : 0.8 ml/L (Arrow shows slight necrosis and degeneration of filaments)

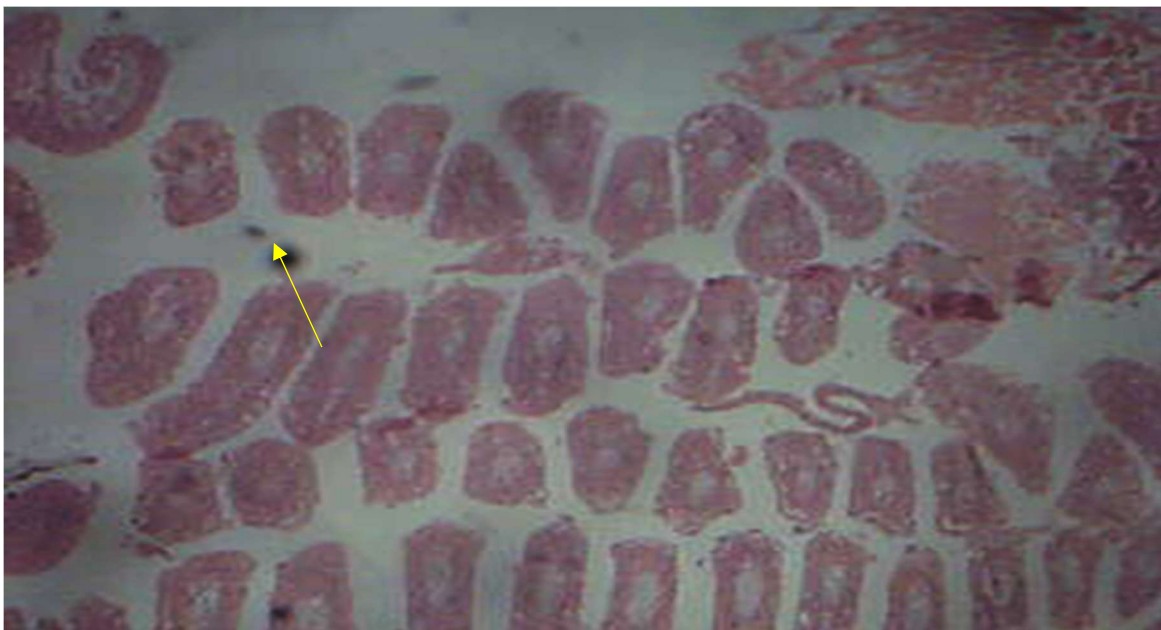


Figure 5: 1.6 ml/L (Arrow shows degeneration of filaments)



**Figure 6:** 2.0 ml/L (Arrow shows severe degeneration of filaments, necrosis and areas of lesion)

#### 4.0 Discussion

The constant exposure of fish to toxic substances leads to high mortality rate in the aquatic ecosystem. Dead fishes were carefully identified by absolute lack of movement and were removed as soon as they were detected. There were no survivors recorded in the highest concentration (2.0ml/L), no mortality was observed in the control tank. Three basic physico-chemical parameters of water were measured before stocking of the fish and within the 96 hours introduction of the toxicant. Dissolved oxygen had a value range of 3.1–3.6, with a temperature of 28°C and a pH range of 5.82–7.19. In fisheries and aquaculture, all these parameters have standard values or acceptable range of values. For dissolved oxygen, a range of 6.0mg/L, for pH a range of 6.7–8.6 and for temperature a range of 25°C–30°C (Ajah, 2007; Smith, 1982; Udo, 2007). The ranges of the physico-chemical parameters of the experimental water were found to fall into the acceptable range before the commencement of the experiment as previously reported by the authors under reference.

In the present study, percentage mortalities were concentration-dependent. The higher the concentration, the higher the percentage mortalities. Similar report was presented by Ogundiran et al. (2010) while investigating the toxicological impacts of detergent effluent in fingerlings of African catfish *Clarias gariepinus* and George et al. (2014) while also investigating the acute toxic effect of Qua Ibo Light Crude oil on the gills of *Clarias gariepinus* juveniles. Calta et al. (2004) when studying the acute toxicity of the synthetic pyrethroid deltamethrin to young mirror carp, *Cyprinus carpio*, Ayotunde et al. (2011) while investigating the toxicity of Carica papaya on adult *C. gariepinus*, Ayuba and Ofojekwu (2002) while investigating on acute toxicity of diazinon to African catfish *Clarias gariepinus*.

In this study, the 96 hours LC<sub>50</sub> of 1.05ml/L obtained from the experimental design is in close range with the LC<sub>50</sub>OF 1.08ml/L obtained during a study on acute toxicity of water-soluble fractions (WSF) of kerosene on Nile tilapia, *Oreochromis niloticus* fingerlings under laboratory condition, undergone by Absalom et al. (2009). The effect of the kerosene observed histopathologically showed a severe destruction of the gill lamellae of *Clarias gariepinus*. However, the gill lamellae of the fish in the control medium (0ml/L) showed no effect on the gill, i.e. the gill lamellae were normal with

no lesion, necrosis and inflammation. In the 0.8ml/L concentration of toxicant it was observed that the gill lamellae of the fish eroded and showed slight necrosis and degeneration filament. In 1.6ml/L concentration of toxicant the gill lamellae of *C. gariepinus* were observed to have shown a degeneration of filament and a destruction of the gill secondary lamellae while in the 2ml/L concentration, it was observed that the gill showed severe degeneration of filaments, necrosis and inflammation; hyperplastic effects was observed on the gill membrane and a loss of gill membrane. These observations correlate with the concentrations of toxicants in each experimental design (aquaria), i.e., the higher the concentration of toxicant, the greater the damages done on the fish gill. These results align with what George et al. (2014) got during their study on the acute toxic effect of Qua Iboe light crude oil on gills of *Clarias gariepinus* juveniles. And also, what Absalom et al. (2009) observed during his studies on the “Acute Toxicity of Water-Soluble Fractions (WSF) of kerosene on Nile Tilapia, *Oreochromis niloticus* fingerlings under Laboratory Conditions.” Hypertrophic, necrotic, atrophy and dystrophy of secondary lamellae have also been reported in haematology and histopathology of *Clarias garpienus* juveniles exposed to refined petroleum oil and kerosene under laboratory conditions (Gabriel et al. 2007).

The changes observed on the gills of *C. gariepnus* falls under the general reactions of fish species organs to toxicant and environmental pollutant, and aquatic pollution. Fernandes and Mazon (2003) observed that fish gills are the prime target organ of all pollutants due to their extensive surface in contact with the external medium and the reduced distance between the external medium, and gill morphology are important biomarkers providing a fast method of detection of the effect of pollutants (Gabriel et al., 2007; George et al., 2014).

The general morphological changes in the gills recorded in this study have been reported by Gabriel et al. (2007) in his study on haematology and gill pathology of *Clarias gariepnus* exposed to petroleum oil and kerosene as a current study by George et al. (2014) on the acute toxic effect of Qua Iboe Light Crude oil on the gills of *C. gariepnus* still reveals same. Fish mortality could also have resulted from direct toxicant of the kerosene. The behavioural observations reported in this experiment have been reported by several other toxicants. Ayuba and Ofojekwu (2002), Svecevicus (2006), George et al. (2014), Absalom et al. (2009), Edori et al. (2014) and Gabriel et al. (2007). Respiratory struggle noticed in exposed fish could be as a result of mucous precipitation on the gill epithelia in response to the toxicant which result in abnormal behaviour as stated by Banerjee (2007).

In fish, direct contact between the aquatic environment and the gill epithelium may cause these surfaces to become sensitive to environmental alteration in the presence of toxic substances and irritants (Absalom et al., 2009). A regular trend of increased mortality was observed with increase I toxicant concentrations showing a dose-response relationship 100% mortality was recorded in the group with the highest concentration of toxicant, i.e. 2.0ml/L, 75% to 1.6ml/L, 70% to 1.2, 10% to 0.8ml/L, 5% to 0.4ml/L and 0% to control group. The mean value of 96-hour LC50 of kerosene to the test fish was calculated to give 1.05ml/L (different LC50 values have been recorded for different contaminants/toxicants, variation in LC50 may also be as a result of differences in type, size, age and strength of the exposed fish and technical grade involved in toxicant preparation) (Palanivela et al., 2005). The dead fish in all the toxicant exposures showed fishes with brownish gills and this corresponds with the findings of Absalom et al. (2009). The plate 1 above shows the distribution of gills in a normal fish control (0ml/L) of kerosene; plate 2 shows the kerosene action on the fish at 0.8ml/L concentration; and plate 3 shows the action of the toxicant to the fish at 1.6ml/L, plate 4 shows the reaction of kerosene (toxicant on the fish gills at 2ml/L which was the highest concentration.

## **5.0 Conclusion and recommendations**

The results of this study demonstrate that kerosene, even at low concentrations, is highly toxic to *C. gariepinus*. Short-term exposure to concentrations exceeding 1.6 ml/L induces significant stress responses in fish, including behavioral changes such as loss of balance, erratic swimming, aggression, weakness, and ultimately high mortality, particularly at higher concentrations due to kerosene-induced stress on the fish's immune system. Histological analysis of the gills revealed vascular congestion, hyperplasia, and loss of the gill membrane, highlighting the toxic effects of kerosene on vital fish organs. As such, there is an urgent need for caution to prevent kerosene contamination in aquatic environments caused by oil refinery activities, pipeline vandalism, accidents, or improper waste disposal. The findings emphasize that kerosene levels in aquatic ecosystems should not exceed 1.05 ml/L, based on the 96-hour LC50 value identified in this experiment. Overall, preventing kerosene contamination is vital for preserving aquatic biodiversity and ensuring the sustainability of aquaculture sector and we recommend that oil companies must adopt ecologically friendly practices for managing spills and waste and adherence to standard waste disposal methods and prompt action during accidental spills.

## **Acknowledgements**

### **Conflicts of Interest**

The authors have no conflicts of interest to declare.

### **Authors' Contribution**

Conceptualization: JEA; Supervision: APE & VE; Resources mobilization: JEA; Data Collection and Investigation: JEA; Materials and Methodology: JEA; Writing the Original Draft Manuscript: JEA, APE & VE; Data Curation, Software, and Formal Analysis: JEA; Visualization and Project Administration: JEA; Validation and Review Editing: JEA, APE & VE. All author reviewed the final manuscript and approved its submission to the journal for publication.

### **Data availability statement**

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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